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Reply to the Editor:

I thank Ma and colleagues for their comments on our article, ''Rat Model of Cardiopulmonary Bypass for Deep Hypothermic Circulatory Arrest.''[1] Ma and colleagues mention that peripheral cannulation alone is not necessarily innovative. I agree, and I would clarify that what we deemed innovative was the combination of: vacuum-assisted venous drainage, a small prime volume membrane oxygenator, nearly normal high-flow delivery rates, peripheral cannulation, ultrasonic flow meters, and active cooling and rewarming with a heat exchanger. In combination, this is a sophisticated cardiopulmonary bypass model for the rat.

These animals can be kept alive beyond bypass in this model without the need for endotracheal positive-pressure ventilation. The flow rates achieved on bypass of 100 to 120 mL/(kg · min) are adequate for rats during hypothermia. Although I agree that ventilation may be easier for survival surgery, it is not a requisite. Its main use in our model is to provide positive pressure to help reexpand the lungs. We have successfully weaned these animals with gradual warming and an oxygen-rich environment without endotracheal intubation.

We routinely administer inotropic medications, as we do in human patients, when separating from cardiopulmonary bypass as a response to low blood pressure and acidemia; these parameters are routinely monitored by means of i-Stat (Abbott Laboratories. Abbott Park, Ill) and indwelling arterial lines in real time during the procedure. I agree that a venous line is useful, and we routinely infuse though the right atrial venous cannula when we are keeping our animals alive. We administer protamine and remove the femoral cannula when animals are being kept alive after bypass, with gentle hemostasis at the exit sites of the catheters. With these modifications, we have established a successful clinically relevant model of cardiopulmonary bypass and deep hypothermic circulatory arrest in the rat.

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SOME ISSUES ABOUT THE DECISION-MAKING PROCESS FOR TRICUSPID VALVE REPAIR

To the Editor:

Congratulations to Roshanali and colleagues[1] for their valuable study, ''Echocardiographic Approach to the Decision-Making Process for Tricuspid Valve Repair,'' which appeared in the June 2010 issue. Functional tricuspid regurgitation develops secondary to the annular dilation as a result of enlargement of the right ventricular cavity, which in turn is related to the increase in pulmonary arterial pressure secondary to left ventricular pathology. This physiopathology does not coincide with the view provided by Roshanali and colleagues' original Figure 1,[1] because elongation of the leaflets and chordae is seen in regurgitation. The absence of coaptation depth among the data given in their original Table 1 supports our claim.[1]

The criterion for pericardial patch use is unclear, although preoperative tricuspid annular diameter is given in their original Table 1.[1] The ring size and the method of reduction of annular diameter are unclear as well. We think that—at least in this study—the correlation between the degree of postoperative tricuspid regurgitation and pulmonary arterial pressure should be investigated.

The number of patients in this study is adequate for such statistical analysis. Reevaluation of the data therefore could yield important results.

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